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NEWS 19 FEB 23 MEDLINE now offers more precise author group fields and 2009 MeSH terms

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=> s (diphtheria toxin receptor) and (process or processing)
9713 DIPHTHERIA
1 DIPHTHERIAS
9714 DIPHTHERIA
(DIPHTHERIA OR DIPHTHERIAS)
89043 TOXIN
94085 TOXINS
137381 TOXIN
(TOXIN OR TOXINS)
793121 RECEPTOR
730830 RECEPTORS
949788 RECEPTOR
(RECEPTOR OR RECEPTORS)
179 DIPHTHERIA TOXIN RECEPTOR
(DIPHTHERIA(W)TOXIN(W)RECEPTOR)
2760848 PROCESS
1897189 PROCESSES
4122776 PROCESS
(PROCESS OR PROCESSES)
636337 PROCESSING
621 PROCESSINGS
636629 PROCESSING
(PROCESSING OR PROCESSINGS)
L1 29 (DIPHTHERIA TOXIN RECEPTOR) AND (PROCESS OR PROCESSING)

=> s L1 and antibody
343609 ANTIBODY
414371 ANTIBODIES
547407 ANTIBODY
(ANTIBODY OR ANTIBODIES)
L2 8 L1 AND ANTIBODY

=> duplicate remove L2
PROCESSING COMPLETED FOR L2
L3 8 DUPLICATE REMOVE L2 (0 DUPLICATES REMOVED)

=> d L3 bib abs 1-8

L3 ANSWER 1 OF 8 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2008:846759 CAPLUS
DN 149:102676
TI Clearance of influenza virus from the lung depends on migratory langerin +CD11b- but not plasmacytoid dendritic cells
AU GeurtsvanKessel, Corine H.; Willart, Monique A. M.; van Rijt, Leonie S.; Muskens, Femke; Kool, Mirjam; Baas, Chantal; Thielemans, Kris; Bennett, Clare; Clausen, Bjoern E.; Hoogsteden, Henk C.; Osterhaus, Albert D. M. E.; Rimmelzwaan, Guus F.; Lambrecht, Bart N.
CS Department of Pulmonary Medicine, Erasmus University Medical Centre Rotterdam, Rotterdam, 3015 GE, Neth.
SO Journal of Experimental Medicine (2008), 205(7), 1621-1634
CODEN: JEMEAV; ISSN: 0022-1007
PB Rockefeller University Press
DT Journal
LA English
AB Although dendritic cells (DCs) play an important role in mediating protection against influenza virus, the precise role of lung DC subsets, such as CD11b- and CD11b+ conventional DCs or plasmacytoid DCs (pDCs), in different lung compartments is currently unknown. Early after intranasal infection, tracheal CD11b-CD11chi DCs migrated to the mediastinal lymph nodes (MLNs), acquiring co-stimulatory mols. in the process. This emigration from the lung was followed by an accumulation of CD11b+CD11chi DCs in the trachea and lung interstitium. In the MLNs, the CD11b+ DCs contained abundant viral nucleoprotein (NP), but these cells failed to present antigen to CD4 or CD8 T cells, whereas resident CD11b-CD8.alpha.+ DCs presented to CD8 cells, and migratory CD11b-CD8.alpha.- DCs presented to CD4 and CD8 T cells. When lung CD11chi DCs and macrophages or langerin+CD11b-CD11chi DCs were depleted using either CD11c-diphtheria toxin receptor (DTR) or langerin-DTR mice, the development of virus-specific CD8+ T cells was severely delayed, which correlated with increased clin. severity and a delayed viral clearance. 120G8+ CD11cint pDCs also accumulated in the lung and LNs carrying viral NP, but in their absence, there was no effect on viral clearance or clin. severity. Rather, in pDC-depleted mice, there was a redn. in antiviral antibody prodn. after lung clearance of the virus. This suggests that multiple DCs are endowed with different tasks in mediating protection against influenza virus.
RE.CNT 63 THERE ARE 63 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2007:568001 CAPLUS
DN 147:8027
TI Mature DC from skin and skin-draining LN retain the ability to acquire and efficiently present targeted antigen
AU Henri, Sandrine; Siret, Carole; Machy, Patrick; Kissenpfennig, Adrien; Malissen, Bernard; Leserman, Lee
CS Centre d'Immunologie de Marseille-Luminy, Faculte des Sciences de Luminy, Aix-Marseille Universite, Marseille, Fr.
SO European Journal of Immunology (2007), 37(5), 1184-1193
CODEN: EJIMAF; ISSN: 0014-2980
PB Wiley-VCH Verlag GmbH & Co. KGaA
DT Journal
LA English
AB Skin-draining LN contain several phenotypically distinguishable DC populations, which may be immature or mature. Mature DC are generally considered to have lost the capacity to acquire and present newly encountered Ag. Using antibody-opsonized liposomes as Ag carriers, we show that mature DC purified from skin explants are able to efficiently capture liposomes, process Ag encapsulated within them and activate Ag-specific CD4+ T cells. Explant DC from mice with Langerhans cells (LC) expressing the primate diphtheria toxin receptor that were exposed to diphtheria toxin in vivo presented Ag as well as explant DC from wild-type mice, indicating that LC are not required and dermal DC are probably responsible for this presentation. We further show that all DC subtypes from LN that capture opsonized Ag are capable of cross-presenting it to CD8+ T cells. Induction of addnl. maturation in vivo by LPS or treatment with double-stranded RNA did not alter the Ag presentation capacity of the skin or LN DC subtypes. These results suggest that mature DC present in skin-draining LN may play an important role in the induction of primary and/or secondary immune responses against Ag delivered to the LN that they take up by receptor-mediated endocytosis.
RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2006:383647 CAPLUS
DN 144:439970
TI Use of fusion proteins that can be taken up by skin cells to deliver therapeutic macromolecules to the bloodstream without injection
IN Mrsny, Randall J.
PA Trinity Biosystems, Inc., USA

SO PCT Int. Appl., 131 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006044205	A2	20060427	WO 2005-US35803	20051004
WO 2006044205	A3	20060908		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

AU 2005296064	A1	20060427	AU 2005-296064	20051004
CA 2583202	A1	20060427	CA 2005-2583202	20051004
US 20060153798	A1	20060713	US 2005-244349	20051004
EP 1804832	A2	20070711	EP 2005-816333	20051004

R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU

CN 101072584	A	20071114	CN 2005-80041667	20051004
JP 2008515808	T	20080515	JP 2007-534904	20051004
IN 2007CN01897	A	20070831	IN 2007-CN1897	20070504
PRAI US 2004-615970P	P	20041004		
US 2005-684484P	P	20050524		
US 2005-718907P	P	20050919		
WO 2005-US35803	W	20051004		

AB Methods of using peptides that promote cellular uptake and transfer of proteins to deliver macromols. to the bloodstream through the skin without the need for injection are described. The protein of interest is applied as a fusion protein with a receptor binding domain, a transcytosis domain, and a cleavable linker. Generally, the cleavable linker is cleavable by an enzyme present in higher concn. at or near the basal-lateral membrane of a polarized epithelial cell or in the plasma than elsewhere in the body, for example, at the apical side of the polarized epithelial cell. The low rate of delivery of the processed protein can also lessen the risk of developing an immune response to the therapeutic protein (no data). In other aspects, the invention provides nucleic acids encoding delivery

constructs of the invention, kits comprising delivery constructs of the invention, cells expressing delivery constructs of the invention, and methods of using delivery constructs of the invention. Expts. developing the system using green fluorescent protein using cell cultures and rat trachea is demonstrated.

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2009 ACS on STN
AN 1994:672962 CAPLUS
DN 121:272962
OREF 121:49627a,49630a
TI Biosynthesis and processing by phorbol ester of the cell surface-associated precursor form of heparin-binding EGF-like growth factor
AU Raab, Gerhard; Higashiyama, Shigeki; Hetelekidis, Stella; Abraham, Judith A.; Damm, Deborah; Ono, Minoru; Klagsbrun, Michael
CS Harvard Med. Sch., Children's Hospital, Boston, MA, 02115, USA
SO Biochemical and Biophysical Research Communications (1994), 204(2), 592-7
CODEN: BBRCA9; ISSN: 0006-291X
PB Academic
DT Journal
LA English
AB Human MDA MB 231 cells were found to synthesize mostly the cell surface-assocd. precursor form of heparin-binding EGF-like growth factor (HB-BGF), a 27-kDa protein. Evidence for this form of HB-EGF included increased fluorescence intensity when cells analyzed by flow cytometry using anti-HB-EGF antibodies, lack of HB-EGF in conditioned medium, and sensitivity to diphtheria toxin, for which HB-EGF is the receptor. Phorbol ester treatment of cells resulted, within 30 min, in loss of cell surface 27 kDA HB-EGF, lack of interaction with anti-HB-EGF antibodies, accumulation of active 21 kDa HB-EGF in conditioned medium, and the acquisition of diphtheria toxin resistance. It was concluded that cell surface-assocd. HB-EGF is the precursor of a bioactive growth factor, is biol. active as the receptor for diphtheria toxin, and is susceptible to rapid processing.

L3 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2009 ACS on STN
AN 1990:401810 CAPLUS
DN 113:1810
OREF 113:375a,378a
TI Localization of the diphtheria toxin receptor -binding domain to the carboxyl-terminal Mr .apprx.6000 region of the toxin
AU Rolf, John M.; Gaudin, Helen M.; Eidels, Leon

CS Southwest. Med. Cent., Univ. Texas, Dallas, TX, 75235-9048, USA
SO Journal of Biological Chemistry (1990), 265(13), 7331-7
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English
AB The C-terminal Mr 5982 peptide of diphtheria toxin was prepd. by specific cleavage of the toxin with hydroxylamine and purified by fast-performance liq. chromatog. The identity of the peptide was established by a combination of SDS PAGE anal., reactivity with specific monoclonal antibodies, and N-terminal sequence anal. The Mr 5982 peptide protected highly toxin-sensitive Vero cells from the lethal action of diphtheria toxin. This protection was due to inhibition of the initial step in the cytotoxic process, the binding of toxin to its receptor. Apparently, the Mr 5982 C-terminal region (amino acid residues 482-535) is, or contains, the receptor-binding domain of diphtheria toxin.

L3 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2009 ACS on STN
AN 1990:50312 CAPLUS
DN 112:50312
OREF 112:8537a,8540a
TI Monoclonal antibodies against Vero cells that protect against diphtheria toxin
AU Ronnberg, Bengt J.; Lidgerding, Burt C.; Middlebrook, John L.
CS Dep. Toxinol., U. S. Army Med. Res. Inst. Infect. Dis., Frederick, MD, 21701, USA
SO Toxicon (1989), 27(10), 1095-104
CODEN: TOXIA6; ISSN: 0041-0101
DT Journal
LA English
AB Mice were immunized with a cell line (Vero) that possesses a high no. of membrane receptors for diphtheria toxin. Spleen cells from these mice were fused with SP2/0-Ag14 cells and 2 cell lines (1A2 and 2D2) isolated by screening for the ability of their secreted antibodies to inhibit binding of radiolabeled diphtheria toxin to Vero cells. These antibodies protected Vero cells from the inhibition of protein synthesis mediated by diphtheria toxin. The antibodies were purified, iodinated, and their binding characteristics investigated. At 4.degree., the assocn. of 1A2 and 2D2 with Vero cells was saturable (KD .apprxeq. 10-8M) and indicated .apprx.106 binding sites/cell. Diphtheria toxin did not inhibit the binding of either radiolabeled antibody . Monoclonal antibody 1A2 completely inhibited 125I-labeled 2D2 binding and vice versa. Trypsin or phospholipase C treatment of Vero cells had no effect on the ability of the monoclonal antibodies to bind to the cells. Apparently, (1) the 2 monoclonal antibodies recognize the same or closely related epitopes and (2) the antibodies bind a domain distinct from the toxin binding site or

to a subcomponent of the diphtheria toxin receptor that is present at many other cell surface sites. These antibodies offer a powerful tool to study the structure, processing, and mode of action of diphtheria toxin receptors.

L3 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2009 ACS on STN
AN 1981:563541 CAPLUS
DN 95:163541
OREF 95:27235a,27238a
TI Effect of ammonium chloride on receptor-mediated uptake of diphtheria toxin by Vero cells
AU Dorland, Rebecca B.; Middlebrook, John L.; Leppla, Stephen H.
CS US Army Med. Res. Inst. Infect. Dis., Frederick, MD, 21701, USA
SO Experimental Cell Research (1981), 134(2), 319-27
CODEN: ECREAL; ISSN: 0014-4827
DT Journal
LA English
AB The mechanism of NH4Cl-mediated protection of Vero cells against diphtheria toxin was studied. In the presence of protective concns. of NH4Cl, Vero cells bound, internalized, and degraded radiolabeled diphtheria toxin at the same rate and to the same extent as did the control cells. However, when specific antibody was added to NH4Cl-treated cells, a fraction of potentially lethal toxin mols. was maintained in a position accessible to antibody neutralization. Apparently, 2 processing mechanisms exists for diphtheria toxin: a nonproductive bulk degrdn. pathway and a productive NH4Cl-sensitive pathway by which active fragment is eventually delivered to the cytoplasm.

L3 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2009 ACS on STN
AN 1979:605403 CAPLUS
DN 91:205403
OREF 91:33007a,33010a
TI Receptor-mediated internalization and degradation of diphtheria toxin by monkey kidney cells
AU Dorland, Rebecca B.; Middlebrook, John L.; Leppla, Stephen H.
CS US Army Med. Res. Inst. Infect. Dis., Fort Detrick, MD, 21701, USA
SO Journal of Biological Chemistry (1979), 254(22), 11337-42
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English
AB The ability of a no. of enzymes and chems. to remove cell surface-bound radiolabeled diphtheria toxin was tested; the combination of pronase [9036-06-0] and inositol hexaphosphate [83-86-3] (PIHP) proved most effective. Using PIHP, the kinetics of toxin-cell assocn. at 37.degree. was resolved into 2 components, surface binding and internalization. The

PIHP assay also allowed estn. of the half-time of toxin internalization (.apprx.25 min). An assay involving pptn. of culture supernatants with trichloroacetic acid was developed and used to measure the rate of degrdn. and excretion of cell-assocd. toxin. Agents which markedly inhibited toxin internalization similarly prevented degrdn., implying an intracellular location for the degradative process. The primary radioactive product excreted by Vero cells was monoiodotyrosine. The extent and rate of toxin degrdn. indicated lysosomal involvement. Finally, agents which blocked internalization or degrdn., or both, (e.g. antibody and concanavalin A [11028-71-0]), protected cells from the cytotoxic action of diphtheria toxin, suggesting that these processes are necessary for expression of the biol. effect.

=> s (pro-HB-EGF) and (process or processing)
95621 PRO
1642 PROS
97229 PRO
(PRO OR PROS)
87688 HB
9935 HBS
90726 HB
(HB OR HBS)
30378 EGF
49 EGFS
30389 EGF
(EGF OR EGFS)
31 PRO-HB-EGF
(PRO(W)HB(W)EGF)
2760848 PROCESS
1897189 PROCESSES
4122776 PROCESS
(PROCESS OR PROCESSES)
636337 PROCESSING
621 PROCESSINGS
636629 PROCESSING
(PROCESSING OR PROCESSINGS)
L4 12 (PRO-HB-EGF) AND (PROCESS OR PROCESSING)

=> s L4 and antibody
343609 ANTIBODY
414371 ANTIBODIES
547407 ANTIBODY
(ANTIBODY OR ANTIBODIES)
L5 2 L4 AND ANTIBODY

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L6 2 DUPLICATE REMOVE L5 (0 DUPLICATES REMOVED)

=> d L6 bib abs 1-2

L6 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2007:920630 CAPLUS
DN 147:421898
TI G protein .beta..gamma. subunits augment UVB-induced apoptosis by stimulating the release of soluble heparin-binding epidermal growth factor from human keratinocytes
AU Seo, MiRan; Lee, Mi-Jeong; Heo, Jin Hee; Lee, Yun-Il; Kim, Yeni; Kim, So-Young; Lee, Eun-So; Juhnn, Yong-Sung
CS Department of Biochemistry and Molecular Biology and Cancer Research Institute, Seoul National University College of Medicine, Seoul, 110-779, S. Korea
SO Journal of Biological Chemistry (2007), 282(34), 24720-24730
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB UV radiation induces various cellular responses by regulating the activity of many UV-responsive enzymes, including MAPKs. The .beta..gamma. subunit of the heterotrimeric GTP-binding protein (G.beta..gamma.) was found to mediate UV-induced p38 activation via epidermal growth factor receptor (EGFR). However, it is not known how G.beta..gamma. mediates the UVB-induced activation of EGFR, and thus we undertook this study to elucidate the mechanism. Treatment of HaCaT-immortalized human keratinocytes with conditioned medium obtained from UVB-irradiated cells induced the phosphorylations of EGFR, p38, and ERK but not that of JNK. Blockade of heparin-binding EGF-like growth factor (HB-EGF) by neutralizing antibody or CRM197 toxin inhibited the UVB-induced activations of EGFR, p38, and ERK in normal human epidermal keratinocytes and in HaCaT cells. Treatment with HB-EGF also activated EGFR, p38, and ERK. UVB radiation stimulated the processing of pro-HB-EGF and increased the secretion of sol. HB-EGF in medium, which was quantified by immunoblotting and protein staining. In addn., treatment with CRM179 toxin blocked UV-induced apoptosis, but HB-EGF augmented this apoptosis. Moreover, UVB-induced apoptosis was reduced by inhibiting EGFR or p38. The overexpression of G.beta.1.gamma.2 increased EGFR-activating activity and sol. HB-EGF content in conditioned medium, but the sequestration of G.beta..gamma. by the carboxyl terminus of G protein-coupled receptor kinase 2 (GRK2ct) produced the opposite effect. The activation of Src increased UVB-induced,

G.beta..gamma.-mediated HB-EGF secretion, but the inhibition of Src blocked that. Overexpression of G.beta..gamma. increased UVB-induced apoptosis, and the overexpression of GRK2ct decreased this apoptosis. We conclude that G.beta..gamma. mediates UVB-induced human keratinocyte apoptosis by augmenting the ectodomain shedding of HB-EGF, which sequentially activates EGFR and p38.

RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2009 ACS on STN

AN 2001:84701 CAPLUS

DN 134:126503

TI Angiotensin AT1 and AT2 receptors differentially regulate angiopoietin-2 and vascular endothelial growth factor expression and angiogenesis by modulating heparin binding-epidermal growth factor (EGF)-mediated EGF receptor transactivation

AU Fujiyama, Soichiro; Matsubara, Hiroaki; Nozawa, Yoshihisa; Maruyama, Katsuya; Mori, Yasukiyo; Tsutsumi, Yoshiaki; Masaki, Hiroya; Uchiyama, Yoko; Koyama, Yoko; Nose, Atsuko; Iba, Osamu; Tateishi, Eriko; Ogata, Nahoko; Jyo, Nobuo; Higashiyama, Shigeki; Iwasaka, Toshiji

CS Dep. Medicine II and Ophthalmology, Kansai Medical Univ., Oska, Japan

SO Circulation Research (2001), 88(1), 22-29
CODEN: CIRUAL; ISSN: 0009-7330

PB Lippincott Williams & Wilkins

DT Journal

LA English

AB Angiotensin II (Ang II)-mediated signals are transmitted via heparin binding epidermal growth factor (EGF)-like growth factor (HB-EGF) release followed by transactivation of EGF receptor (EGFR). Although Ang II and HB-EGF induce angiogenesis, their link to the angiopoietin (Ang)-Tie2 system remains undefined. We tested the effects of Ang II on Ang1, Ang2, or Tie2 expression in cardiac microvascular endothelial cells expressing the Ang II receptors AT1 and AT2. Ang II significantly induced Ang2 mRNA accumulations without affecting Ang1 or Tie2 expression, which was inhibited by protein kinase C inhibitors and by intracellular Ca2+ chelating agents. Ang II transactivated EGFR via AT1 and inhibition of EGFR abolished the induction of Ang2. Ang II caused processing of pro-HB-EGF in a metalloproteinase-dependent manner to stimulate maturation and release of HB-EGF. Neutralizing anti-HB-EGF antibody blocked EGFR phosphorylation by Ang II. Ang II also upregulated vascular endothelial growth factor (VEGF) expression in an HB-EGF/EGFR-dependent manner. AT2 inhibited AT1-mediated Ang2 expression and phosphorylation of EGFR. In an in vivo corneal assay, AT1 induced angiogenesis in an HB-EGF-dependent manner and enhanced the angiogenic activity of VEGF. Although neither

Ang2 nor Ang1 alone induced angiogenesis, sol. Tie2-Fc that binds to angiopoietins attenuated AT1-mediated angiogenesis. These findings suggested that (1) Ang II induces Ang2 and VEGF expression without affecting Ang1 or Tie2 and (2) AT, stimulates processing of pro-HB-EGF by metalloproteinases, and the released HB-EGF transactivates EGFR to induce angiogenesis via the combined effect of Ang2 and VEGF, whereas AT2 attenuates them by blocking EGFR phosphorylation. Thus, Ang II is involved in the VEGF-Ang-Tie2 system via HB-EGF-mediated EGFR transactivation, and this link should be considerable in pathol. conditions in which collateral blood flow is required.

RE.CNT 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s (pro-HB-EGF) and (cleave or cleavage or cleaving)

95621 PRO
1642 PROS
97229 PRO
(PRO OR PROS)
87688 HB
9935 HBS
90726 HB
(HB OR HBS)
30378 EGF
49 EGFS
30389 EGF
(EGF OR EGFS)
31 PRO-HB-EGF
(PRO(W)HB(W)EGF)
12095 CLEAVE
9680 CLEAVES
20829 CLEAVE
(CLEAVE OR CLEAVES)
232484 CLEAVAGE
6114 CLEAVAGES
235383 CLEAVAGE
(CLEAVAGE OR CLEAVAGES)
13202 CLEAVING
2 CLEAVINGS
13203 CLEAVING
(CLEAVING OR CLEAVINGS)

L7 6 (PRO-HB-EGF) AND (CLEAVE OR CLEAVAGE OR CLEAVING)

=> s L7 and antibody

343609 ANTIBODY
414371 ANTIBODIES
547407 ANTIBODY
(ANTIBODY OR ANTIBODIES)
L8 1 L7 AND ANTIBODY

=> d L8 bib abs 1

L8 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2003:389324 CAPLUS
DN 139:115428
TI TACE cleavage of proamphiregulin regulates GPCR-induced proliferation and motility of cancer cells
AU Gschwind, Andreas; Hart, Stefan; Fischer, Oliver M.; Ullrich, Axel
CS Department of Molecular Biology, Max-Planck Institute of Biochemistry, Martinsried, D-82152, Germany
SO EMBO Journal (2003), 22(10), 2411-2421
CODEN: EMJODG; ISSN: 0261-4189
PB Oxford University Press
DT Journal
LA English
AB Communication between G protein-coupled receptor (GPCR) and epidermal growth factor receptor (EGFR) signaling systems involves cell surface proteolysis of EGF-like precursors. The underlying mechanisms of EGFR signal transactivation pathways, however, are largely unknown. We demonstrate that in squamous cell carcinoma cells, stimulation with the GPCR agonists LPA or carbachol specifically results in metalloprotease cleavage and release of amphiregulin (AR). Moreover, AR gene silencing by siRNA or inhibition of AR biol. activity by neutralizing antibodies and heparin prevents GPCR-induced EGFR tyrosine phosphorylation, downstream mitogenic signaling events, cell proliferation, migration and activation of the survival mediator Akt/PKB. Therefore, despite some functional redundancy among EGF family ligands, the present study reveals a distinct and essential role for AR in GPCR-triggered cellular responses. Furthermore, we present evidence that blockade of the metalloprotease-disintegrin tumor necrosis factor-.alpha.-converting enzyme (TACE) by the tissue inhibitor of metalloprotease-3, a dominant-neg. TACE mutant or RNA interference suppresses GPCR-stimulated AR release, EGFR activation and downstream events. Thus, TACE can function as an effector of GPCR-mediated signaling and represents a key element of the cellular receptor cross-talk network.
RE.CNT 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

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=> S (diphtheria toxin receptor) and (cleave or cleavage or cleaving)
        9713 DIPHTHERIA
           1 DIPHTHERIAS
        9714 DIPHTHERIA
             (DIPHTHERIA OR DIPHTHERIAS)
       89043 TOXIN
       94085 TOXINS
      137381 TOXIN
             (TOXIN OR TOXINS)
      793121 RECEPTOR
      730830 RECEPTORS
      949788 RECEPTOR
             (RECEPTOR OR RECEPTORS)
         179 DIPHTHERIA TOXIN RECEPTOR
             (DIPHTHERIA(W)TOXIN(W)RECEPTOR)
       12095 CLEAVE
        9680 CLEAVES
       20829 CLEAVE
             (CLEAVE OR CLEAVES)
      232484 CLEAVAGE
        6114 CLEAVAGES
      235383 CLEAVAGE
             (CLEAVAGE OR CLEAVAGES)
       13202 CLEAVING
           2 CLEAVINGS
       13203 CLEAVING
             (CLEAVING OR CLEAVINGS)
L9           8 (DIPHTHERIA TOXIN RECEPTOR) AND (CLEAVE OR CLEAVAGE OR
CLEAVING)

=> s L9 and antibody
      343609 ANTIBODY
      414371 ANTIBODIES
      547407 ANTIBODY
             (ANTIBODY OR ANTIBODIES)
L10          3 L9 AND ANTIBODY

=> duplicate remove L10
PROCESSING COMPLETED FOR L10
L11          3 DUPLICATE REMOVE L10 (0 DUPLICATES REMOVED)

=> d L11 bib abs 1-3

L11  ANSWER 1 OF 3  CAPLUS  COPYRIGHT 2009 ACS on STN
AN   2006:383647  CAPLUS
DN   144:439970
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TI Use of fusion proteins that can be taken up by skin cells to deliver therapeutic macromolecules to the bloodstream without injection

IN Mrsny, Randall J.

PA Trinity Biosystems, Inc., USA

SO PCT Int. Appl., 131 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006044205	A2	20060427	WO 2005-US35803	20051004
WO 2006044205	A3	20060908		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
AU 2005296064	A1	20060427	AU 2005-296064	20051004
CA 2583202	A1	20060427	CA 2005-2583202	20051004
US 20060153798	A1	20060713	US 2005-244349	20051004
EP 1804832	A2	20070711	EP 2005-816333	20051004
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU				
CN 101072584	A	20071114	CN 2005-80041667	20051004
JP 2008515808	T	20080515	JP 2007-534904	20051004
IN 2007CN01897	A	20070831	IN 2007-CN1897	20070504
PRAI US 2004-615970P	P	20041004		
US 2005-684484P	P	20050524		
US 2005-718907P	P	20050919		
WO 2005-US35803	W	20051004		

AB Methods of using peptides that promote cellular uptake and transfer of proteins to deliver macromols. to the bloodstream through the skin without the need for injection are described. The protein of interest is applied as a fusion protein with a receptor binding domain, a transcytosis domain, and a cleavable linker. Generally, the cleavable linker is cleavable by an enzyme present in higher concn. at or near the basal-lateral membrane of a polarized epithelial cell or in the plasma than elsewhere in the

body, for example, at the apical side of the polarized epithelial cell. The low rate of delivery of the processed protein can also lessen the risk of developing an immune response to the therapeutic protein (no data). In other aspects, the invention provides nucleic acids encoding delivery constructs of the invention, kits comprising delivery constructs of the invention, cells expressing delivery constructs of the invention, and methods of using delivery constructs of the invention. Expts. developing the system using green fluorescent protein using cell cultures and rat trachea is demonstrated.

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2002:937303 CAPLUS
DN 138:20443
TI Endocrine disruptor screening using DNA chips of endocrine disruptor-responsive genes
IN Kondo, Akihiro; Takeda, Takeshi; Mizutani, Shigetoshi; Tsujimoto, Yoshimasa; Takashima, Ryokichi; Enoki, Yuki; Kato, Ikunoshin
PA Takara Bio Inc., Japan
SO Jpn. Kokai Tokkyo Koho, 386 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 2002355079	A	20021210	JP 2002-69354	20020313
PRAI JP 2001-73183	A	20010314		
JP 2001-74993	A	20010315		
JP 2001-102519	A	20010330		

AB A method and kit for detecting endocrine-disrupting chems. using DNA microarrays are claimed. The method comprises prepg. a nucleic acid sample contg. mRNAs or cDNAs originating in cells, tissues, or organisms which have been brought into contact with a sample contg. the endocrine disruptor. The nucleic acid sample is hybridized with DNA microarrays having genes affected by the endocrine disruptor or DNA fragments originating in these genes have been fixed. The results obtained are then compared with the results obtained with the control sample to select the gene affected by the endocrine disruptor. Genes whose expression is altered by tri-Bu tin, 4-octaphenol, 4-nonylphenol, di-N-Bu phthalate, dichlorohexyl phthalate, octachlorostyrene, benzophenone, diethylhexyl phthalate, diethylstilbestrol (DES), and 17-.beta. estradiol (E2), were found in mice by DNA chip anal.

L11 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2009 ACS on STN
AN 1990:401810 CAPLUS
DN 113:1810
OREF 113:375a,378a
TI Localization of the diphtheria toxin receptor -binding domain to the carboxyl-terminal Mr .apprx.6000 region of the toxin
AU Rolf, John M.; Gaudin, Helen M.; Eidels, Leon
CS Southwest. Med. Cent., Univ. Texas, Dallas, TX, 75235-9048, USA
SO Journal of Biological Chemistry (1990), 265(13), 7331-7
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English
AB The C-terminal Mr 5982 peptide of diphtheria toxin was prepd. by specific cleavage of the toxin with hydroxylamine and purified by fast-performance liq. chromatog. The identity of the peptide was established by a combination of SDS PAGE anal., reactivity with specific monoclonal antibodies, and N-terminal sequence anal. The Mr 5982 peptide protected highly toxin-sensitive Vero cells from the lethal action of diphtheria toxin. This protection was due to inhibition of the initial step in the cytotoxic process, the binding of toxin to its receptor. Apparently, the Mr 5982 C-terminal region (amino acid residues 482-535) is, or contains, the receptor-binding domain of diphtheria toxin.